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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
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FULL ESTIMATED COST	1.14	44.06

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FILE LAST UPDATED: 30 Jan 2007 (20070130/ED)

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=> S ADENOSINE A2A AND A1 RECEPTOR
89528 ADENOSINE
766 ADENOSINES

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         89713 ADENOSINE
                 (ADENOSINE OR ADENOSINES)
          2944 A2A
          1172 ADENOSINE A2A
                 (ADENOSINE(W)A2A)
         62116 A1
        684735 RECEPTOR
        628156 RECEPTORS
        815239 RECEPTOR
                 (RECEPTOR OR RECEPTORS)
          3570 A1 RECEPTOR
                 (A1(W)RECEPTOR)
L4         295 ADENOSINE A2A AND A1 RECEPTOR

=> S L4 AND PY<2005
      25013707 PY<2005
L5         239 L4 AND PY<2005

=> S L5 AND PARKINSON?
         25807 PARKINSON?
L6          10 L5 AND PARKINSON?

=> D IBIB ABS HITSTR

L6   ANSWER 1 OF 10  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2004:995771  CAPLUS
DOCUMENT NUMBER:          141:424179
TITLE:                    Imidazolyl benzothiazoles as adenosine receptor
                          ligands, processes for their preparations,
                          pharmaceutical formulations and uses thereof
INVENTOR(S):              Flohr, Alexander; Jakob-Roetne, Roland; Norcross,
                          Roger David; Riemer, Claus
PATENT ASSIGNEE(S):       Hoffmann-La Roche Inc., USA
SOURCE:                   U.S. Pat. Appl. Publ., 37 pp.
                          CODEN: USXXCO
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
     ---------------      ----   --------    --------------------    --------
     US 2004229862         A1    20041118    US 2004-843241          20040511 <--
     US 7122545            B2    20061017
     AU 2004238508         A1    20041125    AU 2004-238508          20040506 <--
     CA 2523959            A1    20041125    CA 2004-2523959         20040506 <--
     WO 2004101558         A1    20041125    WO 2004-EP4843          20040506 <--
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
             AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
             EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
             SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
             SN, TD, TG
     EP 1636217            A1    20060322    EP 2004-731369          20040506
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
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IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK					
BR 2004010315	A	20060523	BR 2004-10315		20040506
CN 1780831	A	20060531	CN 2004-80011781		20040506
JP 2006528215	T	20061214	JP 2006-529752		20040506
PRIORITY APPLN. INFO.:			EP 2003-9842	A	20030513
			WO 2004-EP4843	W	20040506

OTHER SOURCE(S): MARPAT 141:424179
GI

/ Structure 1 in file .gra /

AB Title compds. I [wherein R1 = Ph or N/O-heterocycle; R2 = (un)annulated imidazole, or pharmaceutically acceptable salts thereof] were prepared as adenosine receptor ligands. Also disclosed are the processes for the prepns. of I, pharmaceutical formulations comprising I, and use of I for the treatment of Alzheimer's disease, depression, Parkinson's disease and ADHD. Thus, coupling of imidazole-2-carboxylic acid with 2-methoxy-5-(morpholin-4-yl)phenylamine (9%), followed by treatment with Lawesson reagent (59%), and subsequent cyclization in the presence of potassium hexacyanoferrate (47%) gave compound II. I were measured to have a good affinity to human adenosine A2A receptor and human adenosine A1 receptor with pKi of 7.0-9.3 and 5.1-57, resp.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> D IBIB ABS HITSTR 2-10

L6 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:908902 CAPLUS
DOCUMENT NUMBER: 142:93773
TITLE: Novel Bicyclic Piperazine Derivatives of Triazolotriazine and Triazolopyrimidines as Highly Potent and Selective Adenosine A2A Receptor Antagonists
AUTHOR(S): Peng, Hairuo; Kumaravel, Gnanasambandam; Yao, Gang; Sha, Li; Wang, Joy; Van Vlijmen, Herman; Bohnert, Tonika; Huang, Carol; Vu, Chi B.; Ensinger, Carol L.; Chang, Hexi; Engber, Thomas M.; Whalley, Eric T.; Petter, Russell C.
CORPORATE SOURCE: Departments of Medicinal Chemistry, Pharmacology, and Computational Drug Design, Biogen Idec Inc., Cambridge, MA, 02142, USA
SOURCE: Journal of Medicinal Chemistry (2004), 47(25), 6218-6229
CODEN: JMCMAR; ISSN: 0022-2623
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 142:93773
GI

/ Structure 2 in file .gra /

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AB A series of bicyclic piperazine derivs. of triazolotriazine and triazolopyrimidines was synthesized. Some of these analogs show high affinity and excellent selectivity for adenosine A2a receptor vs. the adenosine A1 receptor. Structure-activity-relationship (SAR) studies based on octahydropyrrolo[1,2-a]pyrazine and octahydropyrido[1,2-a]pyrazine with various capping groups are reported. Among these analogs, the most potent and selective A2a antagonist I [X = N, R = 3-FC6H4] has a Ki value of 0.2 nM and is 16,500-fold selective with respect to the A1 receptor. Among a number of compds. tested, I [X = N, CH, R = H] exhibited significantly improved metabolic stability. These compds. showed good oral efficacy in rodent catalepsy models of Parkinson 's disease.

REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:604078 CAPLUS
DOCUMENT NUMBER: 141:168324
TITLE: Striatal adenosine A2A receptor blockade increases extracellular dopamine release following L-DOPA administration in intact and dopamine-denervated rats
AUTHOR(S): Golembiowska, Krystyna; Dziubina, Anna
CORPORATE SOURCE: Institute of Pharmacology, Polish Academy of Sciences, Krakow, 31343, Pol.
SOURCE: Neuropharmacology (2004), 47(3), 414-426
CODEN: NEPHBW; ISSN: 0028-3908
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The influence of the selective adenosine A2A receptor antagonist ZM 241385 on exogenous L-DOPA-derived dopamine (DA) release in intact and dopamine-denervated rats was studied using an in vivo microdialysis in freely moving animals. Local infusion of L-DOPA (2.5 μM) produced a marked increase in striatal extracellular DA level in intact and malonate-lesioned rats. Intrastriatal perfusion of ZM 241385 (50-100 μM) had no effect on basal extracellular DA level, but enhanced dose-dependently the L-DOPA-induced DA release in intact and malonate-lesioned animals. A non-selective adenosine A2A receptor antagonist DMPX (100 μM), similarly to ZM 241385, accelerated conversion of L-DOPA in intact and malonate-denervated rats. This effect was not produced by the adenosine A1 receptor antagonist, CPX (10-50 μM). However, ZM 241385 did not affect the L-DOPA-induced DA release in rats pretreated with reserpine (5 mg/kg i.p.) and α-methyl-p-tyrosine (AMPT, 300 mg/kg i.p.). Obtained results indicate that blockade of striatal adenosine A2A receptors increases the L-DOPA-derived DA release possibly by indirect mechanism exerted on DA terminals, an effect dependent on striatal tyrosine hydroxylase activity. Selective antagonists of adenosine A2A receptors may exert a beneficial effect at early stages of Parkinson's disease by enhancing the therapeutic efficacy of L-DOPA applied exogenously.

REFERENCE COUNT: 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:563751 CAPLUS
DOCUMENT NUMBER: 141:167237

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TITLE: Piperazine Derivatives of [1,2,4]Triazolo[1,5-a][1,3,5]triazine as Potent and Selective Adenosine A2a Receptor Antagonists

AUTHOR(S): Vu, Chi B.; Peng, Bo; Kumaravel, Gnanasambandam; Smits, Glenn; Jin, Xiaowei; Phadke, Deepali; Engber, Thomas; Huang, Carol; Reilly, Jennifer; Tam, Stacy; Grant, Donna; Hetu, Gregg; Chen, Liqing; Zhang, Jianbo; Petter, Russell C.

CORPORATE SOURCE: Department of Medicinal Chemistry, Biogen Idec Inc., Cambridge, MA, 02142, USA

SOURCE: Journal of Medicinal Chemistry (2004), 47(17), 4291-4299
CODEN: JMCMAR; ISSN: 0022-2623

PUBLISHER: American Chemical Society

DOCUMENT TYPE: Journal

LANGUAGE: English

OTHER SOURCE(S): CASREACT 141:167237

AB The [1,2,4]triazolo[1,5-a]triazine derivative 3, more commonly known in the field of adenosine research as ZM-241385, has previously been demonstrated to be a potent and selective adenosine A2a receptor antagonist, although with limited oral bioavailability. This [1,2,4]triazolo[1,5-a]triazine core structure has now been improved by incorporating various piperazine derivs. With some preliminary optimization, the A2a binding affinity of some of the best piperazine derivs. is almost as good as that of compound 3. The selectivity level over the adenosine A1 receptor subtype for some of the more active analogs is also fairly high, >400-fold in some cases. Many compds. within this piperazine series of [1,2,4]triazolo[1,5-a]triazine have now been shown to have good oral bioavailability in the rat, with some as high as 89% (compound 35). More significantly, some piperazines derivs. of [1,2,4]triazolo[1,5-a]triazine also possessed good oral efficacy in rodent models of Parkinson's disease. For instance, compound 34 was orally active in the rat catalepsy model at 3 mg/kg. In the 6-hydroxydopamine-lesioned rat model, this compound was also quite effective, with a min. ED of 3 mg/kg po.

REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:226605 CAPLUS

TITLE: Evaluation of analogues of (-)-mefloquine as adenosine A2A receptor antagonists

AUTHOR(S): Gillespie, Roger J.; Giles, Paul R.; Lerpiniere, Joanne; Ward, Simon E.; Weiss, Scott M.; Knight, Tony R.; Misra, Anil; Benwell, Karen; Dourish, Colin T.; Cliffe, Ian A.

CORPORATE SOURCE: Vernalis Research Ltd, Winnersh, RG41 5UA, UK

SOURCE: Abstracts of Papers, 227th ACS National Meeting, Anaheim, CA, United States, March 28-April 1, 2004 (2004), MEDI-247. American Chemical Society: Washington, D. C.
CODEN: 69FGKM

DOCUMENT TYPE: Conference; Meeting Abstract

LANGUAGE: English

AB Evaluation of analogs of (-)-mefloquine as adenosine A2A receptor antagonists. The adenosine A2A receptor plays an important role in regulating smooth and well-coordinated movement, in part, by modulating the activity of dopamine sensitive neurons in the striatum. Blockade of the adenosine A2A receptor has been shown to offer considerable promise as a novel treatment

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for the symptoms of Parkinson's disease. We have discovered that (-)-(11R,2'S)-α-2-piperidinyl-2,8-bis(trifluoromethyl)-4-quinolinemethanol, the (-)-enantiomer of the antimalarial drug mefloquine, is a potent and moderately selective adenosine A2A receptor antagonist. This compound has a Ki of 61 nM at human adenosine A2A receptors, is moderately selective over human adenosine A1 receptors (Ki 255 nM), and highly selective over human adenosine A2B and A3 receptors (Ki 7072 and 6941 nM, resp.). The synthesis and evaluation of a series of analogs of mefloquine as adenosine A2A antagonists will be described.
(-)-mefloquine.

L6 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:855799 CAPLUS
DOCUMENT NUMBER: 139:350637
TITLE: Preparation of 5-oxo and 5-thio derivatives of 5H-indeno[1,2-b]pyridine with adenosine A2a receptor binding and phosphodiesterase inhibiting activity for the treatment of neurodegenerative disorders and inflammation related diseases
INVENTOR(S): Heintzelman, Geoffrey R.; Averill, Kristin M.; Dodd, John H.; Demarest, Keith T.; Tang, Yuting; Jackson, Paul F.
PATENT ASSIGNEE(S): Ortho-McNeil Pharmaceutical, Inc., USA
SOURCE: PCT Int. Appl., 112 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE	
WO 2003088963	A1	20031030	WO 2002-US30825		20020927	<--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW						
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG						
US 2003212089	A1	20031113	US 2002-123389		20020416	<--
US 6958328	B2	20051025				
CA 2488929	A1	20031030	CA 2002-2488929		20020927	<--
AU 2002341875	A1	20031103	AU 2002-341875		20020927	<--
BR 2002015699	A	20050503	BR 2002-15699		20020927	
CN 1809349	A	20060726	CN 2002-810472		20020927	
PRIORITY APPLN. INFO.:			US 2002-123389	A	20020416	
			US 2001-284465P	P	20010418	
			WO 2002-US30825	W	20020927	

OTHER SOURCE(S): MARPAT 139:350637
GI

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* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB The title compds. [I; R1 = COR5 (wherein R5 = H, alkyl, aryl, arylalkyl), CO2R6 (R6 = H, alkyl, aryl, arylalkyl), CN, etc.; R2 = alkyl, aryl, heteroaryl, etc.; R3 = H, halo, alkyl, etc.; R4 = H, alkyl, CH2Ph, etc.; X = S, O], useful for treating disorders ameliorated by antagonizing adenosine A2a receptors or reducing PDE activity in appropriate cells, were prepared Thus, oxidation of dihydropyridine II (preparation given) afforded 81% III. The IC50 and %inhibition data on PDE 4,5 and 7A, and Ki on A2a and A1 receptors binding for representative compds. I were given. Pharmaceutical compns. comprising the compound I are claimed. This invention also provides therapeutic and prophylactic methods using the instant pharmaceutical compns.
REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:119391 CAPLUS
DOCUMENT NUMBER: 138:363047
TITLE: Adenosine receptor blockade reverses hypophagia and enhances locomotor activity of dopamine-deficient mice
AUTHOR(S): Kim, Douglas S.; Palmiter, Richard D.
CORPORATE SOURCE: Molecular and Cellular Biology Program, University of Washington, Seattle, WA, 98195-7275, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2003), 100(3), 1346-1351
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Adenosine receptors modulate dopaminergic function by regulating dopamine release in presynaptic neurons and intracellular signaling in postsynaptic striatal neurons. To investigate how adenosine impinges on the action of dopamine in feeding and locomotion, genetically altered, dopamine-deficient mice were treated with adenosine receptor antagonists. Acute administration of the nonselective adenosine receptor antagonist, caffeine (5-25 mg/kg i.p.), reversed the hypophagia of mutant mice and induced hyperactivity in both control and mutant animals. However, caffeine treatment elicited much less hyperactivity in dopamine-deficient mice than did L-3,4-dihydroxyphenylalanine (L-DOPA) administration, which partially restores dopamine content. Caffeine treatment enhanced feeding of L-DOPA-treated mutants but, unexpectedly, it reduced their hyperlocomotion. Caffeine administration induced c-Fos expression in the cortex of dopamine-deficient mice but had no effect in the striatum by itself. Caffeine attenuated dopamine agonist-induced striatal c-Fos expression. An antagonist selective for adenosine A2A receptors induced feeding and locomotion in mutants much more effectively than an A1 receptor antagonist. L-DOPA-elicited feeding and hyperlocomotion were reduced in mutants treated with an A1 receptor agonist, whereas an A2A receptor agonist decreased L-DOPA-induced feeding without affecting locomotion. The observations suggest that the hypophagia and hypoactivity of mutants result not only because of the absence of dopamine but also because of the presence of A2A receptor signaling. This study of a genetic model of dopamine depletion provides evidence that A2A receptor antagonists could ameliorate the hypokinetic symptoms of advanced Parkinson's disease patients without inducing excessive motor activity.
REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS

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50613257

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:891652 CAPLUS
DOCUMENT NUMBER: 138:301232
TITLE: Neuroprotective role of adenosine in the CNS
AUTHOR(S): Wardas, Jadwiga
CORPORATE SOURCE: Department of Neuropsychopharmacology, Institute of Pharmacology, Polish Academy of Sciences, Krakow, PL 31-343, Pol.
SOURCE: Polish Journal of Pharmacology (2002), 54(4), 313-326
CODEN: PJPAE3; ISSN: 1230-6002
PUBLISHER: Polish Academy of Sciences, Institute of Pharmacology
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. It is well established that in the CNS, endogenous adenosine plays a pivotal role in neurodegeneration. A low, nanomolar concentration of adenosine is normally present in the extracellular fluid, but it increases dramatically during enhanced nerve activity, hypoxia or ischemia. In these pathol. conditions, adenosinergic transmission-potentiating agents, which elevate adenosine level by either inhibiting its degradation (adenosine deaminase and kinase inhibitors) or preventing its transport, offer protection against ischemic or excitotoxic neuronal damage. The directly acting adenosine A1 receptor agonists are known to mediate neuroprotection, mostly by the blockade of Ca2+ influx, which results in the inhibition of glutamate release and reduction of its excitatory effects at a postsynaptic level. More recent data have shown that antagonists of adenosine A2A receptors markedly reduce cerebral ischemic damage in animal models of focal and global ischemia. Moreover, these compds. attenuate the neuronal loss induced by excitatory amino acids (EAA). A neuroprotective effect of adenosine A2A receptor antagonists was also shown in animal models of Parkinson's disease (MPTP, 6-OHDA, methamphetamine). Hence, it might be suggested that adenosine A2A receptor antagonists may represent a novel strategy in the therapeutic approach to pathologies characterized by acute or chronic neurodegenerative events, since they not only reverse motor impairment but can act as neuroprotective compds. by promoting cell survival.
REFERENCE COUNT: 117 THERE ARE 117 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1998:644563 CAPLUS
DOCUMENT NUMBER: 130:33316
TITLE: Adenosine A2A receptors modify motor function in MPTP-treated common marmosets
AUTHOR(S): Kanda, Tomoyuki; Tashiro, Tomomi; Kuwana, Yoshihisa; Jenner, Peter
CORPORATE SOURCE: Pharmaceutical Research Institute, Kyowa Hakko Kogyo Co Ltd, Shizuoka, 411-8731, Japan
SOURCE: NeuroReport (1998), 9(12), 2857-2860
CODEN: NERPEZ; ISSN: 0959-4965
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Both adenosine A1 and A2 receptor populations are located in the striatum and can modify locomotor activity, and they may form a therapeutic target for Parkinson's disease (PD). Administration of the selective

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adenosine A2A antagonist (E)-1,3-diethyl-8-(3,4-dimethoxystyryl)-7-methyl-3,7-dihydro-1H-purine-2,6-dione (KW-6002) to MPTP-treated common marmosets increased locomotor activity. In contrast, administration of the selective A1 receptor antagonist 1,3-dipropyl-8-cyclopentylxantine (DPCPX) had no effect on locomotion. Administration of the adenosine A2A receptor agonist 2-[p-[2-(2-aminoethylamino) carbonylethyl] phenethyl amino]-5'-N-ethylcarboxamidoadenosine (APEC) dose dependently suppressed basal locomotor activity. A minimally ED of APEC (0.62 mg/kg, i.p) completely reversed the increase in locomotor activity produced by administration of KW-6002. The adenosine A2A receptor appears to be an important target for the treatment of basal ganglia disorders, particularly PD.

REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1996:360396 CAPLUS
DOCUMENT NUMBER: 125:26394
TITLE: Adenosine A2 receptor-mediated excitatory actions on the nervous system
AUTHOR(S): Sebastiao, A. M.; Ribeiro, J. A.
CORPORATE SOURCE: Laboratory of Pharmacology, Gulbenkian Institute of Science, Oeiras, 2781, Port.
SOURCE: Progress in Neurobiology (Oxford) (1996), 48(3), 167-189
CODEN: PGNBA5; ISSN: 0301-0082
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with >200 refs. The distribution, mol. structure and role of adenosine A2 receptors in the nervous system, is reviewed. The adenosine A2a receptor subtype, identified in the nervous system with ligand binding, functional studies or genetic mol. techniques, has been demonstrated in the striatum and other basal ganglia structures, in the hippocampus, in the cerebral cortex, in the nucleus tractus solitarius, in motor nerve terminals, in noradrenergic terminals in the vas deferens, in myenteric neurons of the ileum, in the retina and in the carotid body. The A2b receptors have been identified in glial and neuronal cells, and may have a widespread distribution in the brain. Activation of adenosine A2a receptors can enhance the release of several neurotransmitters, such as acetylcholine, glutamate, and noradrenaline. The release of GABA might be either enhanced or inhibited by A2a receptor activation. The A2 receptor activation also modulates neuronal excitability, synaptic plasticity, as well as locomotor activity and behavior. The ability of A2 receptors to interact with other receptors for neurotransmitters/neuromodulators, such as dopamine D2 and D1 receptors, adenosine A1 receptors, CGRP receptors, metabotropic glutamate receptors and nicotinic autofacilitatory receptors, expands the range of possibilities used by adenosine to interfere with neuronal function and communication. These A2 receptor-mediated adenosine actions might have potential therapeutic interest, in particular in movement disorders such as Parkinson's disease and Huntington's chorea, as well as in schizophrenia, Alzheimer's disease, myasthenia gravis and myasthenic syndromes.

=> FIL STNGUIDE
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FULL ESTIMATED COST	41.77	85.83

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
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CA SUBSCRIBER PRICE	0.00	-15.60

STN INTERNATIONAL LOGOFF AT 11:07:41 ON 31 JAN 2007

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